given, while his stools increased in number and became more liquid. His blood chlorids that day was 782 milligrams. The child's condition continued to become progressively worse in spite of all treatment, and he died on January 21, 1932.

His autopsy findings were negative except for pus, with very little bony change in the left mastoid antrum.

* * *

CASE 3.—P. G., age five weeks. Admitted to hospital on January 18, 1932, as a boarder. Physical examination and past history were negative. Child was breast fed until date of admission. Weight at that time was eight pounds five ounces. The child was placed on a formula consisting of equal parts of milk and water with some karo. The child did fairly well on the above formula, having gained five ounces in weight, until January 25, 1932. He had been retaining all his feedings and his stools were normal in number, color, and consistency.

On January 26, 1932, the stools increased in number, became greenish in color and of a semi-liquid consistency. His weight dropped and he also began to vomit an occasional feeding. The formula was changed, but the child's condition was progressively becoming worse. The vomiting increased, his stools became more frequent and more liquid. The temperature did not rise until January 30, 1932, when it rose to 101 degrees. Examination on January 29 revealed reddened drums, and on the next day the drums were beginning to bulge, so they were opened and pus obtained from both sides. The temperature dropped after the incision of the drums, but the child did not improve. He became extremely dehydrated, began to vomit all feedings, his stools were very frequent and watery. He died on the evening of January 31, 1932. No autopsy was performed, but a postmortem examination of the mastoids revealed some pus, with a slight amount of bony changes.

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DEPRESSED FRACTURES OF THE ZYGOMA

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FRACTURES of the zygoma are often overlooked and, as they should be treated early, the cardinal points involved will be briefly discussed.

The zygoma has four processes: frontosphenoidal, temporal, orbital, and maxillary. The temporal process is long, narrow and thin, and forms an arch when united posteriorly with the zygomatic process of the temporal bone. Fracture occurs chiefly through the temporal, frontal or orbital processes, the body of the bone then being depressed.

Practically all cases are due to trauma such as automobile or airplane accidents, fist-fights, or falls.

The chief signs are: flatness of the cheek eminence, often anesthesia of the upper lip, trismus due to pressure on the coronoid process of the mandible, blood from the nose if injury to the antrum has occurred, and sometimes when the arch is broken a distinct dimple is visible. X-rays are of great value, especially when there is much swelling, the Watters position, as used for the maxillary sinuses, being the best to show this bone.

The treatment is early surgical care. In most early cases excellent results can be obtained by means of the following intra-oral operation. An incision, two centimeters long, is made in the bucco-alveolar fold above the upper last two molars; then by blunt dissection a flat, broad elevator is placed beneath the beginning of the temporal arch and the bone raised upward and outward. Iodoform gauze is used for drainage and if packed rather tightly may aid in holding the bone in its new position.

In some cases it may be necessary to enter the antrum by way of the canine fossa and then raise the bone.

External operations are done by many. In some cases a combination of the two methods can be used. The more commonly used external operations will be briefly described. A screw porte is forced through the skin into the outer surface of the bone and the fracture reduced by upward and outward elevation. Another method is to use an instrument shaped like a large, heavy towel clip forceps, putting one blade on the orbital process and the other through the skin into the outer surface of the bone and then raising the bone to place. For depressions of the arch some make an incision along the border of the arch and raise the depressed process by use of blunt elevators. Occasionally a heavy aneurysm needle can be passed around the beginning of the arch and the fracture reduced.

Good end-results are obtained in practically all cases where reduction is done early.

1136 West Sixth Street.

QUARTAN MALARIA *

REPORT OF CASE

TREATMENT WITH QUINIDIN SULPHATE IN A PATIENT HYPERSENSITIVE TO QUININ

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AND
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QUARTAN malaria and hypersensitiveness to quinin, both quite uncommon conditions, have recently been observed in the same individual.

QUARTAN MALARIA—OCCURRENCE

Infection by *Plasmodium malariæ* is quite uncommon in all parts of the world, and particularly so in California. Only one report of the disease in this state [1] has appeared in the literature although a moderate number of cases have been observed. In large series of cases of malaria seen by Craig [2] and Thayer and Hewetson,[3] infections by the quartan parasite have been noted in only one of five or six hundred cases. The disease is said by Bass [4] to be prevalent in certain areas of the United States, as in northeastern Louisiana, but there is no definite substantiating evidence of this on record. From published reports, the Indian Medical Service [5,6,7] has encountered quartan infection more frequently than any other group of observers.

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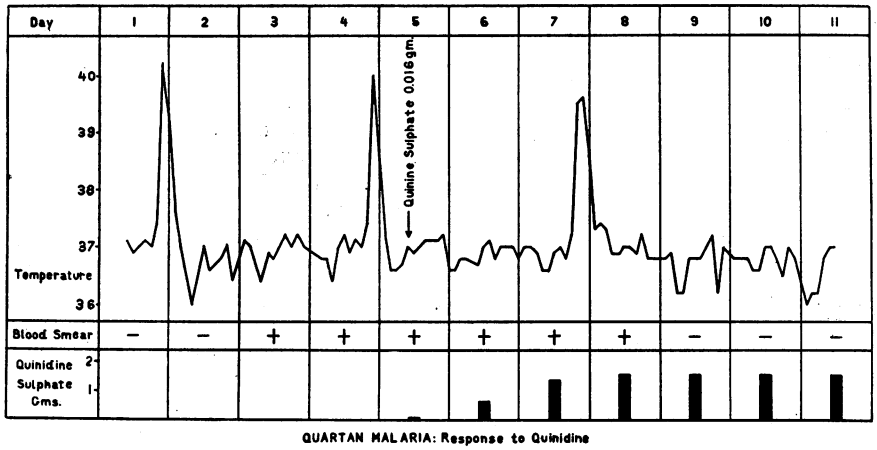

Fig. 1.—Chart showing response to quinidin.

THERAPEUTIC AGENTS IN MALARIA

South American Indians seem to have been familiar with the medicinal value of cinchona bark for many centuries. The recorded use of cinchona derivatives, however, dates from about 1638[8] when cinchona was introduced into Europe by the early explorers. Used first in all fevers, it was found to be especially effective in chills and fever occurring in definite cycles, that is, in malaria. Quinin was soon noted to be the most readily prepared and one of the most effective constituents of cinchona and has achieved almost universal use in malaria. It is known to be curative of malaria when given in effective doses over sufficient time.

Because of unpleasant side effects of quinin and occasional intolerance to the drug, substitutes have long been sought. In view of their effects on spiral organisms particularly, many arsenic preparations have been tried. Their beneficial effect in malaria, if any, was shown to be only slight and temporary. The dyes have been studied, methylene blue in particular having been used in many cases. Here, again, when any effect was noted it was minimal.

Renewed interest in the component alkaloids of cinchona other than quinin was aroused in the second decade of this century. Some fifteen alkaloids of varying toxicity to man and to plasmodia have been investigated. Quinin, cinchonin, cinchonidin, ethylhydrocuprein, and quinidin were studied most extensively. Each of these was found to be therapeutically effective in malaria, quinin and quinidin being most satisfactory and of about equal value.[5,6,7,9,10]

During the past ten years the German workers[11] have been active in the preparation of synthetic substitutes for quinin. Their efforts culminated in the production of plasmochin, a quinolin derivative, which has been shown by use to be less effective than quinin and much more toxic.[12,13,15]

SENSITIZATION TO QUININ

In the ordinary use of quinin there occur unpleasant side actions designated by the general term "cinchonism." In a few individuals an entirely different kind of reaction is noted: nausea, vomiting, headache, dyspnea, coryza, and urticarial manifestations following rapidly on the administration of even small doses of the drug. These reactions are of the nature of allergic responses and are found only in persons who are hypersensitive to quinin. A skin test devised by Boerner in 1917[16] is said to be prophetic. The hypersensitiveness to quinin has been shown by several well-studied cases,[17,18] to extend often to the other levorotatory alkaloids of cinchona, whereas their dextrorotatory isomers[10,19,20] could be administered without ill effect. This is especially striking in the case of quinin and quinidin. In practically all of the reported cases the hypersensitiveness is acquired, *i. e.*, sensitization has occurred, after the use of quinin.[20,21] Local sensitization, as of the skin,[22] without hypersensitiveness to general administration has been noted. Attempts at desensitization have been claimed by some observers to be successful,[23,24,25] but have failed in most hands.

REPORT OF CASE

On February 16, 1932, a single American salesman of thirty-three years entered Lane Hospital, with a complaint of chills and fever for ten and one-half months. The family history is important in that intolerance to quinin is not present in the other members of the family. The patient had traveled widely in the United States, Europe, Africa, Asia, Philippine Islands, and South America from 1920 to 1925, but has been in the western United States since then.

He was perfectly well until one evening in April, 1931, when he was seized suddenly with a severe chill followed by fever of some twelve hours' duration. The chill and fever recurred four days later and every three days thereafter, with two exceptions, until entry into the hospital. During July, 1931, the seizures occurred daily for about thirty days. In January, 1932, there was a period of sixteen days of freedom from seizures. During the past year he had lost forty pounds in weight.

From experience with various cold remedies, the patient knew himself to be intolerant to quinin. However, at the behest of physicians he took quinin in May, 1931, and again in September, 1931. Each taking of the drug was followed by a severe general reaction, characterized by nausea, vomiting, generalized itching and erythema, lacrimation, tinnitus, and photophobia lasting several hours. After these experiences the patient carefully avoided further quinin medication. In December, 1931, he was given five intravenous injections of neoarsphenamin without appreciable effect upon the attacks.

Except for obvious weight loss and a moderately enlarged spleen, the physical examination was negative. The blood showed 4,300,000 red cells, 78 per cent hemoglobin (Sahli), 8,250 leukocytes with 66 per cent polymorphonuclear neutrophils and 30 per cent lymphocytes. The blood Wassermann, blood cultures, and microscopic agglutination of the patient's serum with meningococcus antigen were negative. Urine and stool examinations revealed nothing to aid in the diagnosis.

On the evening of entry the patient had a characteristic malarial paroxysm with malaise followed after thirty minutes by a severe chill with fever to 40.2 degrees centigrade (R). His temperature remained

above normal for nine hours. Seventy-two hours later this course of events was repeated.

Repeated blood studies on the first two days of hospital admission revealed no malarial parasites. On the third day, however, typical quartan parasites were found in small numbers.

On February 19 an unsuccessful attempt was made to produce a skin reaction to quinin sulphate 1:100,000 solution. On the following day, February 20, a test dose of 0.015 grams of quinin sulphate was given by mouth, resulting fifteen minutes later in itching of the scalp. The itching soon became generalized. Within twenty minutes the patient was nauseated and vomited repeatedly, a generalized erythema with chemosis of the conjunctivae and marked lacrimation developed. Stroking of the skin produced a typical urticarial response. An injection of 0.5 cubic centimeter of 1:1000 adrenalin hydrochlorid solution brought about marked relief within five minutes. The original symptoms recurred, however, within half an hour, to persist, with gradual amelioration, for two hours longer. Four hours after taking the quinin the patient felt quite well. Later that day he was given 0.067 grams of quinidin sulphate in divided doses without event. On the following day he received 0.65 grams of the same drug. Thereafter, for four days he was given 1.6 grams of the same drug, quinidin sulphate, daily in four doses. On the third day of quinidin administration the anticipated chill and fever occurred, with only slight alteration (see chart). On the second day after this chill the parasites disappeared from the blood. No further chills occurred. For thirty days after dismissal from the hospital on February 26, 1932, the patient received 1.2 grams of quinidin sulphate daily. To date, June 1, 1932, there has been no recurrence of symptoms, and there has been a gain of thirty pounds in weight.

COMMENT

This case is of particular interest in that it shows: First, the absence of familial hypersensitiveness to quinin; second, the lack of sensitiveness to quinidin in an individual extremely sensitive even to a minute dose of quinin; and third, the efficiency of quinidin in the treatment of quartan malaria.

SUMMARY

1. *Plasmodium malariæ* infection is relatively rare.

2. Hypersensitiveness to quinin, which is usually not inherent but due to sensitization, is uncommon.

3. Sensitization to levorotatory cinchona alkaloids does not usually extend to their dextrorotatory isomers.

4. The occurrence of quartan malaria in an individual hypersensitive to quinin is herewith reported.

5. Quinidin sulphate is effective in the treatment of quartan infection.

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SPIROCHETA PALLIDA—ITS IDENTIFICATION*

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THE darkfield procedure remains the most accurate method for identification of syphilis in its earliest stages.

Improvement of any part of this procedure to allow for a more accurate appraisal seems of distinct value.

The following drawing represents a very simple device having as its major principle, suction. Such devices have been suggested before, yet their common usage has apparently not been adopted.

A five or ten cubic centimeter Luer syringe is attached to a centrifuge tube by a short piece of

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